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Office of Administrative Law Judges
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Issue Date: 09 March 2006

Case No. 2004-BLA-40

In the Matter of:
REBA GARDNER, Survivor of
DANIEL GARDNER, Deceased,
Claimant,

v.

REBEL COAL CO., INC.,
Employer,

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS,
Party in Interest.

APPEARANCES:
Steven A. Sanders, Esq.
On behalf of Claimant

John T. Chafin, Esq.
On behalf of Respondent

BEFORE: THOMAS F. PHALEN, JR.
Administrative Law Judge

DECISION AND ORDER – DENIAL OF BENEFITS

This is a decision and order arising out of a claim for benefits under Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended by the Black Lung Benefits Act of 1977, 30 U.S.C. §§ 901-962, ("the Act") and the regulations thereunder, located in Title 20 of the Code of Federal Regulations. Regulation section numbers mentioned in this Decision and Order refer to sections of that Title.[1]

[1] The Department of Labor amended the regulations implementing the Federal Coal Mine Health and Safety Act of 1969, as amended. These regulations became effective on January 19, 2001, and are found at 65 Fed. Reg. 80, 045-80,107 (2000)(to be codified at 20 C.F.R. Parts 718, 722, 725 and 726). On August 9, 2001, the United States District Court for the District of Columbia issued a Memorandum and Order upholding the validity of the new regulations. All citations to the regulations, unless otherwise noted, refer to the amended regulations.

On December 10, 2003, this case was received at the Office of Administrative Law Judges from the Director, Office of Workers' Compensation Programs, for a hearing. (DX 49).[2] A formal hearing on this matter was held on September 22, 2004, in Prestonsburg, Kentucky by the undersigned Administrative Law Judge. All parties were afforded the opportunity to call and to examine and cross examine witnesses, and to present evidence, as provided in the Act and the above referenced regulations.

ISSUES[3]

The issues in this case are:

1. Whether the Miner had pneumoconiosis as defined by the Act;

2. Whether the Miner's pneumoconiosis arose out of coal mine employment; and

3. Whether the Miner's death was due to pneumoconiosis.

(DX 49; Tr. 10-11).

Based upon a thorough analysis of the entire record in this case, with due consideration accorded to the arguments of the parties, applicable statutory provisions, regulations, and relevant case law, I hereby make the following:

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Background

Daniel Gardner ("Miner") was born on April 8, 1928, and died on June 25, 2000 at the age of 72. (DX 1, 10). On May 4, 1971, Miner married Reba (Hatfield) Gardner ("Claimant"), and they remained married and living together until his death. (DX 1, 11, 39-52; Tr. 13). Claimant's and Miner's applications for benefits both noted that they had a dependent child between the ages of 18 and 23 who was attending school. (DX 1-2, 39-50). Miner, however, submitted a statement noting that his son, Dan, completed high school in June 2000, but as of August 2000, he had not enrolled in another school, and may not for another full year. (DX 9). On June 17, 2002, the Mayo State Vocational Technical School stated that Miner and Claimant's son had attended full-time from May 23, 2001 through January 21, 2002, but as of June 2002, he was no longer attending full-time. (DX 46). Employer does not contest Claimant's eligibility as Miner's survivor, and I find that the evidence supports this designation.

[2] In this Decision, "DX" refers to the Director's Exhibits, "EX" refers to the Employer's Exhibits, "CX" refers to the Claimant's Exhibits, and "Tr." refers to the official transcript of this proceeding

[3] At the hearing, Employer withdrew as uncontested the following issues: whether the person upon whose death the claim is based is a miner; whether the claimant is an eligible survivor of a miner; whether the named employer is the responsible operator; and whether the miner's most recent period of cumulative employment of not less than one year was with the named responsible operator. (Tr. 10-11). The parties also stipulated to 13 years of coal mine employment. (Tr. 10). Total disability was marked on the designation form, but is not an issue in this survivor's claim. §718.205(c).

On his application for benefits, Miner alleged that he engaged in coal mine employment for 15 years. (DX 1). Miner last worked as a blaster, mechanic, and lubeman. (DX 4, 39). He explained that these duties required that he stand for seven hours per day, lift and carry 50 to 100 pounds 9 to 10 times per day, and carry it a distance of 20 to 25 feet. (DX 39-54). Miner also worked in and around coal mines until 1986, when he quit due to closure of the mines. (DX 1.

Claimant submitted an affidavit of Miner's condition. (DX 11). Based on coughing and breathing difficulty for the seven to eight years preceding death, she contended that he suffered from black lung disease. At the hearing, Claimant testified that Miner did not have any trouble breathing when he started working as a coal miner, but toward the end he developed problems, including shortness of breath upon exertion. (Tr. 15-16). She explained that this condition progressed to the point that he could no longer play with his children or cut the grass. (Tr. 16-17). Miner was ultimately admitted into the Parkview Manor nursing home where he spent the last year of his life. (Tr. 20-21). Prior to admission into Parkview Manor, his treating physicians were Drs. Reddy and Jurich, but after admission, he came under the care of Dr. Baretta Casey. (Tr. 21). Claimant stated that Dr. Casey treated her husband for his breathing condition and that this included continual oxygen treatment. (Tr. 21-22).

Procedural History

Miner filed his initial claim for benefits under the Act on November 2, 1995. (DX 39-76). On April 8, 1996, the District Director, Office of Workers' Compensation issued an initial denial of benefits, finding that Miner had failed to prove any of the elements of entitlement. (DX 39-13). Miner did not respond to the Director's letter, so on June 19, 2002 the Director issued a letter stating that the claim was administratively closed due to abandonment. (DX 39-2). Miner took no further action on this claim.

Miner filed a subsequent Claim for benefits on May 25, 2005. (DX 1). Miner, however, died on June 25, 2000. (DX 2). On August 17, 2000, Claimant filed a statement that she wished to continue her husband's claim. (DX 3). Claimant also filed a survivor's claim on August 22, 2000. (DX 2). On September 12, 2000, the District Director denied the living miner claim, finding that Claimant had failed to prove any elements of entitlement. (DX 14). On November 28, 2000, the Director issued a denial of the survivor's claim finding that Claimant had failed to prove any elements of entitlement. (DX 15). On June 4, 2001, the Director issued a letter stating that it had not been able to schedule an informal conference, but that upon reconsideration, it affirmed the previous survivor's claim denial. (DX 31). On June 11, 2001Claimant requested that an informal conference be held to narrow the issues. (DX 34). Both the living miner and survivor's claims were forwarded to the Office of Administrative Law Judges on August 20, 2001 for a formal hearing. (DX 40). On August 27, 2001, Claimant requested the claim be remanded, and as Employer offered no objection, on October 4, 2001 Administrative Law Judge Roketenetz remanded the claim. (DX 42-44, 46). On August 5, 2002, Director issued a memorandum of conference but had not released the decision to the parties by August 15, 2003 when he issued a supplemental memorandum of informal conference denying benefits. Claimant

timely appealed, and on December 10, 2003, this matter was transferred to the Office of Administrative Law Judges for a formal hearing. (DX 49).[4]

Length of Coal Mine Employment

The Social Security Earnings records and the other evidence of record establishes, and I find, that Miner was a coal miner within the meaning of § 402(d) of the Act and § 725.202 of the regulations. The parties stipulated that Miner engaged in at least 13 years of coal mine employment. (Tr. 10). Since the parties' stipulation is supported by the record, (DX 4-6, 39). I find that Miner engaged in at least 13 years of coal mine employment.

Miner's last employment was in the Commonwealth of Kentucky (DX 7-8); therefore, the law of the Sixth Circuit is controlling. [5]

Responsible Operator

Liability under the Act is assessed against the most recent operator that meets the requirements of §§ 725.494 and 725.495. Since Miner's employment with Big Bird Coal Co. in 1986 lasted for less than one year full year, the District Director identified Miner's prior employer, Rebel Coal Co., Inc., as the punitive responsible operator. (DX 7). Rebel Coal Co., Inc., does not contest this issue. (Tr. 10-11). After review of the record, I find that Rebel Coal Co., Inc., is properly designated as the responsible operator in this case.

MEDICAL EVIDENCE

Under §725.2 (c), the limitations of §725.310 of the amended regulations do not apply if a claim was pending before January 19, 2001. Claimant originally filed her claim in 2000. As a result, this claim was filed before the amended regulations, and thus, the evidentiary limitations do not apply.

[4] The Memorandum to File dated December 10, 2003 notes that only the August 17, 2000 claim was transferred to the Office of Administrative Law Judges. (DX 50). It also notes that Miner's November 2, 1995 claim is included in the Director's exhibits but that it is administratively closed and not subject adjudication. This memorandum makes no mention of the living miner's claim filed on May 25, 2000. The August 20, 2001 Memorandum to File, however, transferred both the August 17, 2000 survivor's claim and the May 25, 2000 living miner's claim to the Office of Administrative Law Judges. (DX 41). The Director has provided no explanation as to what happened to the living miner's claim after remand, and thus, it is unclear whether the instant action is a survivor's claim or a combined survivor/living miner claim. However, since neither of the parties mentioned the living miner claim at the hearing or in their briefs, I find that the instant adjudication only includes the August 17, 2000 survivor's claim.

[5] Appellate jurisdiction with a federal circuit court of appeals lies in the circuit where the miner last engaged in coal mine employment, regardless of the location of the responsible operator. *Shupe v. Director, OWCP*, 12 B.L.R. 1-200 (1989)(en banc).

X-RAYS

Exhibit	Date of X-ray	Date of Reading	Physician / Credentials	Interpretation
DX 39-36	02/28/96	2/28/96	West, BCR[6], B-reader[7]	Negative
DX 39-37	02/28/96	3/19/96	Sargent, BCR, B-reader	Quality only
DX 39-6	02/28/96	5/06/96	Gogineni	Negative
DX 39-10	02/28/96	5/07/96	Abramowitz	Negative
DX 39-7	02/28/96	5/08/96	Wershba	Negative
DX 29	10/19/98	1/15/01	Binns	0/1 st
DX 29	10/19/98	1/17/01	Gogineni	0/1 st
DX 29	10/19/98	1/18/01	Baek	0/1 st

PULMONARY FUNCTION TESTS

Exhibit/ Date	Co-op./ Undst./ Tracings	Age/ Height	FEV_1	FVC	MVV	FEV_1/ FVC	Qualifying Results
DX 33 6/10/91	Poor/Good/ Good/ Yes[8]	63 65”	1.02 .51	2.21 1.13	----	46 45	Invalid[9]
DX 39-49 2/28/96	Good/ Good-Fair Yes	67 65”	1.1	1.5	31.2	73	Invalid[10]

[6] A physician who has been certified in radiology or diagnostic roentgenology by the American Board of Radiology, Inc., or the American Osteopathic Association. *See* 20 C.F.R. § 727.206(b)(2)(III). The qualifications of physicians are a matter of public record at the National Institute of Occupational Safety and Health reviewing facility at Morgantown, West Virginia.

[7] A “B” reader is a physician who has demonstrated proficiency in assessing and classifying x-ray evidence of pneumoconiosis by successful completion of an examination conducted by or on behalf of the Department of Health and Human Services. This is a matter of public record at HHS National Institute for Occupational Safety and Health reviewing facility at Morgantown, West Virginia. (42 C.F.R. § 37.5l) Consequently, greater weight is given to a diagnosis by a "B" Reader. *See Blackburn v. Director, OWCP*, 2 B.L.R. 1-153 (1979).

[8] Only one tracing was included with this report.

[9] The report notes poor initial effort requiring the back-extrapolated volume of point zero to be too large. As a result, the reporting physician noted that the FEV1 and FEV3 may be invalid. The second test, however, reported good effort. Both studies appear to be pre-bronchodilator. Dr. N.K. Burki also submitted a validation report finding the study to be unacceptable. (DX 38). Dr. Burki stated that there were insufficient numbers of MVV and FEV tracings, and the effort was less than optimal. Drs. Fino and Broudy also found this study to be invalid. (EX 2-3).

[10] Dr. Fritzhand invalidated this study, noting that Miner was very weak and elderly and that the study was conducted with the Miner lying on a gurney. Drs. Fino and Broudy also found this study to be invalid. (EX 2-3).

ARTERIAL BLOOD GAS STUDIES

Exhibit	Date	pCO_2	pO_2	Qualifying
DX 39-38	2/28/96	37.2	69.3	No
DX 13	4/04/00	44.7	50.7	Yes[11]
DX 13	5/14/00	41.3	129.5	No[12]
DX 13	6/21/00	56	100	Yes[13]
DX 13	6/21/00	34.9	167.3	No[14]
DX 13	6/21/00	52.4	100.1	Yes[15]
DX 13	6/22/00	39.5	78.6	No[16]
DX 13	6/22/00	38.4	168.5	No[17]
DX 13	6/23/00	36.8	75.2	No[18]

Hospital and Treatment Records

The record contains treatment notes from Pikeville Methodist Hospital of Kentucky. (DX 13). These records span from 1998 through 2000, and the entries pertinent to this claim for benefits are reproduced below in chronological order.[19]

November 19, 1998 – X-ray report by Dr. Halbert: The heart is enlarged and the lung fields are very congested in appearance. There appears to be large bilateral effusions which are obscuring both diaphragms. Impression: findings consistent with congestive failure with larger bilateral effusions.

January 13, 1999 – X-ray report by Dr. West: Compared to November 19, 1998, there is some improvement. Heart is enlarged. There are some parenchymal densities in the left lateral lung periphery and the region above the dome of the right diaphragm look stable. There are no new consolidations. Impression. Volume overload with effusions or CHF, but improved.

January 13, 1999 – Examination report by Dr. Shapiro: Patient presents with respiratory difficulties and is being evaluated for upper respiratory tract infection. On examination he has general rhonchi and diffuse expiratory wheezes, bibasilar rales are present. X-ray identified mild

[11] Test conducted on 100% oxygen.
[12] Test conducted on 4 liters nasal cannula O2.
[13] Test conducted on 100% oxygen.
[14] Test conducted on 100% oxygen.
[15] Test conducted on 100% oxygen.
[16] Test conducted on 70% oxygen.
[17] Test conducted on 100% oxygen.
[18] Test conducted on 80% oxygen.
[19] This treatment record includes several x-ray interpretations. With exception of Dr. West, there is no evidence in the record as to the x-ray reading credentials of the providing physicians. Also, these interpretations were related to the treatment of Miner's condition, and not conducted for the purpose of determining the existence or extent of pneumoconiosis. Finally, there is no record of the film quality for any of these x-rays. As a result, the treatment x-ray interpretations are not in compliance with the quality standards of §718.102 and Appendix A to Part 718, and will not be considered under § 718.202(a)(1).

CHF with mildly increased rate, pleural effusion versus baseline. Diagnosis: CHF and bronchitis.

June 7, 1999 – X-ray report by Dr. Kendall: There is a round opacity within the left lung base and chronic pleural parenchymal scarring present bilaterally. There is cardiomegaly present with pulmonary venous hypertension. Impression: Rounded opacity may represent developing infiltrate or possible mass.

June 20, 1999 – X-ray report by Dr. Kendall: There are bilateral pleural effusions present, left greater than right, unchanged from prior exam. There is also bibasilar atelectasis versus infiltrate present which is also unchanged. There is cardiomegaly present with mild pulmonary venous hypertension. Impression: Cardiomegaly with pulmonary venous hypertension and no change in bilateral pleural effusions with bibasilar atelectasis versus infiltrate.

October 1, 1999 – X-ray report by Dr. Halbert: There are infiltrates again seen in the lower lung zones laterally. This appears to be increasing on the right which has a more rounded, fluffy appearance. Patient probably has bilateral effusions and his heart appears to be enlarged. Impression: Interval worsening with increasing infiltrates and/or effusions in the bases.

November 5, 1999 – X-ray report by Dr. Halbert: There are bibasilar infiltrates and effusions that are obscuring the heart margins and diaphragm. No significant change since October 1, 1999.

November 17, 1999 – X-ray report by Dr. Poulos: Compared to November 5, 1999, there is no gross interval change. Findings are compatible with congestive heart failure, infiltrate seen in both lower lung zones and the presence of bilateral pleural effusions. The degree of disease remains stable. Cardiomegaly and atherosclerotic change of the aortic arch is noted. Impression: no change in the degree of infiltration or congestive heart failure noted in both lungs.

March 9, 2000 – X-ray report by Dr. West: The right lung has almost completely cleared and the left lung is improved compared to November 5, 1999. Both costophrenic angles are mildly blunted and there is a persistent density near the left mid chest wall. Heart is enlarged.

March 23, 2000 – X-ray report by Dr. West: Density in the lower lungs that is a little worse than the March 9, 2000 study, but considerably better than the November 17, 1999 film, and equal or better than all the others since November 1998. Possible mild decompression in a patient with chronic CHF. Effusions are minimal with no definite consolidations.

April 4, 2000 – Emergency department admission report by Dr. Hamm: Patient presented with marked labored respirations and hypoxia. History reveals positive for left CVA, peripheral vascular disease with carotid stenosis, TIAs, dementia, periodic aphasia with esophageal dysmotility, speech defect, ataxia, history of hypertension, ASHF, history of bronchitis and bronchial spasms, end stage renal disease secondary to diabetic nephropathy, history of pericardiectomy due to pericardial effusion, chronic anemia, retinopathy, peripheral neuropathy, gastropathy, chronic protein malnutrition, obesity, gastritis, duodenitis, weight loss, iron deficiency, anemia and episodes of hyper anhypokalemia. Examination revealed some wet rales

bilaterally, left greater than right with poor air exchange. ABG showed PCO2 44 and PO2 50. EKG revealed abnormalities. X-ray is positive for extensive pulmonary edema with bilateral effusion. Admitting diagnosis: CHF, bilateral effusions, rule out sepsis, and hyperkalemia.

April 4, 2000 – X-ray report by Dr. Kendall: There is cardiomegly present with diffuse bilateral infiltrates. This may be secondary to edema or pneumonia.

April 4, 2000 – ABG report (see chart above)

April 4, 2000 – Evaluation by Dr. Bhagrath: Examination revealed poor inspiratory effort and breath sounds diminished in both bases. A few rhonchi are present but no wheezes. Chest x-ray predialysis revealed large bilateral pleural effusions and pulmonary congestion. Post dialysis, the pleural effusions persist but are decreased. The pulmonary congestion has also improved but there is some underlying scarring. EKG and ABG were conducted. Impression: Congestive heart failure with bilateral pleural effusions accompanied with severe respiratory failure due to volume overload; hyperkalemia in a patient with end stage renal disease on chronic maintenance hemodialysis; COPD; atherosclerotic heart disease with related cardiomyopathy and chronic left bundle branch block; multi-infarct dementia; insulin requiring diabetes mellitus with multiple end organ complications including severe peripheral vascular disease, status post bilateral below knee amputations; and end state renal disease secondary to diabetic nephropathy on chronic maintenance hemodialysis three times a week.

April 5, 2000 – X-ray report by Dr. West: Bilateral effusions and vascular congestion continue similar to 7:25 am today. Underlying pulmonary infiltrates are not entirely excludable on portable radiography. Impression: CHF with or without infiltrates.

April 27, 2000 – X-ray report by Dr. Kendall: Compared to April 4, 2000, there has been interval resolution of the pulmonary edema. There is mild bibasilar atelectasis present and cardiomegaly with small bilateral pleural effusions.

May 14, 2000 – Examination report by Dr. Villavicencio: Physical examination revealed decreased breath sounds in the bases. ABG study was conducted. Diagnosis: Hypotension, fever, rule out sepsis; chronic renal failure; and elevated cardiac enzymes, rule out false positive values as a result of renal failure. Patient was admitted.

May 14, 2000 – X-ray report by Dr. West: There is no significant change compared to April 27, 2000. There continues to be scattered densities and costophrenic angle blunting in the lower left lung and a somewhat ill-defined diaphragm at the right. The upper lungs continue clear and the heart is enlarged.

May 14, 2000 – ABG study (see chart above).

May 15, 2000 – Examination report by Miner's primary physician, Dr. Casey: Lungs are clear to auscultation. Upon review of laboratory studies, including ABG, EKG, blood cultures, history, and symptoms, Dr. Casey diagnosed condition as a hypotensive episode; UTI, possible sepsis due to fever; end-stage renal disease with chronic hemodialysis.

May 15, 2000 – Discharge summary by Dr. Casey: Patient was admitted and monitored for 23 hours with no change in condition. Felt that he would be treated just as easily at the nursing home. Repeat EKG showed similar changes as the day before with no true acute changes. AMI profile and remained essentially the same with no evidence of myocardial infarction. Diagnosis: urinary tract infection; hypotensive episode resolved; end state renal disease on hemodialysis; hypoproteinemia; congestive heart failure; chronic bronchitis atherosclerotic heart disease; non-insulin-dependent mellitus; diabetic neuropathy, retinopathy, nephropathy, and diabetic gastroparesis; essential hypertension; carotid artery disease; degenerative osteoarthritis; anemia of chronic disease; iron deficiency anemia; atrophic gastritis; cerebrovascular disease; organic brain syndrome; insulin-requiring diabetes; bilateral below-the-knee amputations due to vascular disease; and peripheral vascular disease.

June 21, 2000 – Admission report by Dr. Von Dippe: Patient was brought to emergency room with decreased level of consciousness. Lungs reveal coarse rales and rhonchi in all fields. Portable chest shows lower lobe infiltrate. ABG shows severe hypoxia, large AA gradient with longstanding CO2 retention and partial metabolic compensation. Assessment: Left lower lobe pneumonia with altered level of consciousness, probably bacteremic. Primary physician admitted for further treatment and evaluation.

June 21, 2000 – X-ray report by Dr. Poulos: Compared to the May 13, 2000 study, findings compatible with chronic congestive heart failure. There continues to be increased density at the left lung base obscuring the left hemidiaphragm from infiltration or pleural disease. No definite new pathology is demonstrated.

June 21, 2000 – 3 ABG studies (see chart above)

June 21, 2000 – Consultation report by Dr. Bhagrath: After yesterday's scheduled hemodialysis treatment patient returned to the nursing home where he reportedly had relative hypotension accompanied with some decrease in level of consciousness. This morning he was found unresponsive and finger stick revealed low glucose level. EKG revealed a chronic left bundle branch block with artial fibrillation. A chest x-ray suggested possible left lower lobe pneumonia, although the patient has had bilateral chronic pleural effusions. Physical examination revealed air entry and chest wall excursion equal bilaterally, no rhonchi or wheezes, and a few coarse crackles present in both lung bases. There is some diminished air entry in the bases. Dr. Bhagrath also reviewed ABG values. Impression and Recommendation: end stage renal disease on chronic maintenance hemodialysis three times a week; currently no evidence of acute congestive heart failure or volume overload; electrolytes are satisfactory except for hypenatremia with serum sodium of 150; currently there is no need for acute dialysis; chronic left bundle branch block; hypoglycemic coma; insulin-requiring diabetes mellitus with severe end organ complications; and anemia of chronic renal failure.

June 22, 2000 – Admission diagnosis by Dr. Casey: Family ordered no code. Examination revealed course rhonchi and rales in all fields especially in the left lower lobe. EKG, ABG on 100% oxygen, chest x-ray, and a CT scan of the head were reviewed. Impression: new cerebral cardiovascular accident; respiratory distress with hypoxia and unresponsiveness; COPD;

hypoglycemia in a non-insulin-dependent diabetic insulin requiring; and end stage renal disease on chronic hemodyialysis. Patient is admitted for supportive care but is on a complete do not resuscitate order.

June 23, 2000 – ABG study (see chart above).

June 25, 2000 – Discharge Summary by Dr. Casey: After do not resuscitate order was written, telemetry was discontinued. Multiple orders were written for comfort measures. Patient remained completely unresponsive throughout hospital course. CT scan of head revealed new areas of ischemic infarction involving the right cerebellar hemisphere as well as his old area of infarction involving the left occipital lobe. This was a massive area of stroke. Sputum and blood culture showed nonhemolytic Staph. Hemoccult stools were positive. CBC showed toxic granulation with 26 bands and a white count 6.9. Patient expired on June 25, 2000 and an autopsy was ordered. Discharge diagnosis: Respiratory distress with loss of consciousness and severe hypoglycemic reaction secondary to a large, right, cerebellar hemisphere acute cerebrovascular accident; lower left lobe pneumonia; non-insulin dependent diabetes mellitus; end-stage renal disease on chronic hemodialysis; COPD; history of remote cerebrovascular accident; status post bilateral below the knee amputation carotid artery stenosis; recurrent transient ischemic attack; senile dementia with element of multi-infarct dementia; gastroesophageal reflux disease with periodic dysphagia and espophageal dysmotility; essential hypertension; anterosclerotic heart disease; past history of pericarditis with pericardial effusion; chronic anemia; diabetes mellitus not requiring Insulin with diabetic retinopathy, neuropathy, and nephropathy; diabetic gastroparesis; chronic protein malnutrition; chronic gastritis; history of duodenitis; history of pyloric channel polyps with ulceration; history of positive PPD; history of hypoxalemia; iron deficiency anemia; history of congestive heart failure; and pneumonia Methicillin resistant Staph aureus.

Death Certificate

The death certificate, signed by Dr. Barretta Casey on August 7, 2000, lists that Miner's death was due to COPD, mild bilateral pneumoconiosis, non-insulin dependent diabetes mellitus, and end stated renal disease. (DX 10). Dr. Casey also noted atherosclerotic heart disease as another significant contribution to death.

Autopsy Report

Dr. Sheila Combs performed Miner's autopsy June 27, 2000. (DX 12, CX 2). On gross examination, Dr. Combs noted that the pleural surface of the lungs showed moderate amounts of speckled dark pigmentations. On cut surface, Dr. Combs noted that the bronchi and pulmonary vessels were patent and mildly dilated, but there was no definite evidence of consolidation or centrilobular emphysema. She also noted that the parenchyma was gray-tan and contained a moderate amount of anthracotic pigmentation. On microscopic examination, Dr. Combs identified evidence of chronic airway disease. She also found increased numbers of mucus secreting cells predominance present in bronchioles, and noted that the alveolar septi were mildly thickened and contained increased numbers of macrophages, many of which were laden with black pigment compatible with carbon particles, and some that were compatible with

hemosidering. These macrophages were present within alveolar spaces as well as focal instissated mucus. Dr. Combs next found that the small arteries of the lungs revealed evidence of mild medial hypertrophy and mild intimal fibrosis. She also identified evidence of resolving pneumonia present as a mononuclear rich exudates filling alveolar spaces, and small foci of polymorphonuclear leukocytes that were present within a few bronchiotes, especially in the left lower lobe.

Based on her gross and microscopic descriptions, Dr. Combs diagnosed a history of recent, mild, bilateral pneumonia; mild, bilateral anthracosis; and moderate coronary artery atherosclerosis. She opined that Miner suffered from severely compromised respiratory function, primarily due to chronic airway disease and anthracosis, but that he also had multiple medical problems which greatly contributed to his death. Dr. Combs ultimately concluded that the cause of death was indeterminate due to limitations of the autopsy.[20] She further explained that the limited necropsy cannot conclusively explain Miner's death, but she opined that the overlying pneumonia in this setting would be a contributory factor in his demise.

Narrative Medical Opinion

Dr. Martin Fritzhand examined the Claimant on February 28, 1996. (DX 39: 41). Dr. Fritzhand considered the following: symptomatology (chronic productive cough and occasional pedal edema), employment history (13 years coal mine employment in a strip mine, working as a coal shooter and equipment greaser, last working in 1980), individual history (arthritis, heart disease, diabetes mellitus, high blood pressure, two strokes, chronic renal failure, and multiple hospitalizations), family history (no significant findings), smoking history (smoked three to four cigars per day from 1940 until 1943), physical examination (clear breath sounds without rales, rhonchi, or wheezes), chest x-ray (negative), PFT (invalid), ABG, and an EKG (abnormal). Dr. Fritzhand diagnosed ASHD caused by high blood pressure or possible junctional rhythm. He opined that Miner did not suffer from a pulmonary impairment.

Dr. Echols Hansbarger, a pathologist, submitted a report on September 6, 2000, in which he reviewed Miner's medical records, death certificate, and 14 autopsy slides. (DX 22). On microscopic examinations, Dr. Hansbarger identified extensive acute pneumonitis with polys seen in alveolar spaces, moderate centrilobular emphysema with enlarged airspaces and clubbed septa, and focal deposits of anthracotic pigment without reactive fibrosis that were scattered throughout the lung parenchyma. Dr. Hansbarger specifically noted that coal maculae were not identified. Looking to the bronchial lymph nodes, however, Dr. Hansbarger noted pigment deposition with reactive fibrosis. Based on these observations, Dr. Hansbarger diagnosed extensive, bilateral acute pneumonitis; severe antherosclerotic coronary heart disease; severe cardiac hypertrophy; moderate centrilobular emphysema of the lung; mild, focal anthracotic pigmentation of the lung; and anthracosilicosis of the bronchial lymph nodes. Considering all the evidence he reviewed, Dr. Hansbarger opined that Miner died as a result of acute bronchopneumonia with severe atherosclerotic coronary artery disease, cardiac hypertrophy, chronic renal failure, COPD, and diabetes. He also concluded that there was no evidence of CWP or respiratory impairment caused by coal dust exposure, and as a result, occupational

[20] Claimant granted permission for a limited chest-only autopsy.

exposure could not have contributed or hastened death in any way. Dr. Hansbarger, however, reiterated that this opinion was based solely on the absence of CWP.

Dr. Hansbarger was deposed on November 15, 2000. (DX 27). Dr. Hansbarger reiterated that while he found evidence of anthracotic pigment deposition, there was no evidence of scarring or focal emphysema, and no evidence of coal maculae in Miner's lungs. (DX 27:9). He explained,

> The diagnosis of coal workers' pneumoconiosis needs the findings in the lung itself of … coal maculae. These are collections of anthracotic pigment which is surrounded by scar tissue, that is, the lung's reaction to the anthracotic pigment or coal dust and the formation of some destruction of lung tissue about this coal macule called focal emphysema…. [Y]ou must have these structures within the lung tissue itself in order to make the diagnosis of coal workers' pneumoconiosis.

(DX 27:8). While the remainder of the deposition was generally a repeat of the findings of his earlier written report, Dr. Hansbarger added that Miner suffered some pulmonary disability from his COPD and heart disease, but he again emphasized that Miner had no pulmonary impairment due to his occupational exposure history. (DX 27:11).

Dr. Joshua A. Perper, a clinical professor of pathology, epidemiology and public health, submitted a medical evidence review on November 16, 2000. (DX 26). This review included an independent evaluation of the 14 autopsy slides. On microscopic evaluation of the lung tissue, Dr. Perper identified the following: rare, focal, very slight fibro-anthracotic thickening of pleura; the bronchi show shedding of epithelium, a slight to moderate sub-mucosal chronic inflammatory infiltrate, and in places increased number of mucus-producing cells; in the areas of bronchopneumonia the bronchi and alveoli are filled with acute inflammatory cells; centrilobular emphysema of moderate severity, focal; areas of acute bronchopneumonia; congestion and edema; in places the alveoli contain a mixed inflammatory infiltrate; many of the alveoli contain desquamated cells and clusters of macrophages containing black anthracotic pigment, in some the aveoli contain macrophages with brown-yellowish pigment (hemosiderin); anthracotic deposition, slight in peri-vascular and peri-bronchial areas, none reaching dimensions of macules, and no pneumoniotic nodules present or no birefringent silica crystals observed under polarized light; areas of interstitial fibrosis; and peribronchial lymph nodes show dense anthracosis, but no significant fibrosis or birefringent silica crystals. On microscopic evaluation of the heart, Dr. Perper found the following: Trans-mural patchy fibrosis; focal myocardial fibrosis in the area of the septum; and focal hypertrophy of myocardial fibers. Based on these microscopic findings, Dr. Perper diagnosed acute bronchopneumonia; chronic bronchitis; centrilobular emphysema with interstitial fibrosis, moderately severe, focal; congestion and edema of the lungs; anthracotic pigmentation of pulmonary parenchyma, non-diagnostic, negative for CWP with absence of macules, pneumoconiotic nodules and silica crystals; sclerosis of small intra-pulmonary vessels consistent with pulmonary hypertension; old myocardial infarction; focal myocardial fibrosis; hypertrophy of myocardial fibers, focal; and coronary arteriosclerosis with slight to moderate luminal narrowing.

In addition to his microscopic review and diagnosis, Dr. Perper also considered Dr. Combs' autopsy report, Miner's death certificate, Dr. Hansbarger's medical report, and the Pikeville Hospital records. Dr. Perper did not have any information concerning Miner's occupational exposure. Based on this evidence Dr. Perper concluded that it was not likely that Miner had an occupational lung disease which was caused by his coal mine employment. He reasoned that the gross and microscopic pathological evidence do not substantiate the presence of CWP because anthracotic pigmentation of the pulmonary parenchyma in the absence of pneumoconiotic lesions (macules and nodules) is not sufficient for diagnosing CWP. He explained that the microscopic examination did not show the presence of birefringent silica crystals, which is not diagnostic of pneumoconiosis, but is a marker for exposure to mixed coal dust containing silica or rock dust. Dr. Perper also based this conclusion on the fact that none of the numerous chest x-ray readings noted the presence of pneumoconiotic nodules or opacities. Next, he opined that the clinical finings indicate that Miner's respiratory dysfunction was primarily due to congestive heart failure with a contribution from his chronic bronchitis and COPD. Dr. Perper noted that with exception of the death certificate, virtually none of the hospital medical records included a diagnosis of CWP.

Turning to respiratory impairment at the time of death, Dr. Perper opined that Miner death was the combined result of an unrelated catastrophic cerebellar infarction, congestive arteriosclerotic cardiovascular disease with an extremely large heart, unrelated COPD, and acute terminal bronchopneumonia. He also explained that Miner's death was not significantly contributed to or hastened by pneumoconiosis based on the fact that he did not have evidence of CWP as determined by pathological and x-ray findings. He opined that death was caused by the sudden onset of a deep coma caused by a catastrophic CVA and complicated by bronchopneumonia in a patient with a plethora of pathological conditions, including diabetic nephropathy and renal failure, hypertensive and arterioslcerotic cardiovascular disease, and occupationally unrelated COPD.

Dr. Casey, a family practioner, submitted a letter on March 20, 2001. (DX 33, CX 3). Dr. Casey stated that Miner had been under her care at the Parkview Manor Nursing Home. She explained that at the time of Miner's death, he suffered from multiple medical problems including COPD secondary to CWP, non-insulin dependent diabetes mellitus, and end-stated renal disease secondary to diabetes. She further explained that he died of respiratory failure, which was a direct result of his CWP. Dr. Casey also stated that due to Miner's black lung disease, he was susceptible to multiple lung infections at the time of his death, and that he had had a recent Methicillin resistant Staphylococcus aureus pneumonia. Dr. Casey attached a copy of Miner's death certificate and Dr. Combs' autopsy report to this letter.

Dr. Raphael Caffrey submitted a pathology consultation on September 16, 2001. (DX 45). In addition to the 14 autopsy slides, Dr. Caffrey also considered most of the medical evidence of record. On microscopic examination of the right lung, Dr. Caffrey noted multiple areas that showed either acute pneumonia or resolving pneumonia; a section of main stem bronchus that showed inflammatory cells along the mucosal surface with erosion of the epithelium; lymph node tissue that showed a moderate amount of anthracotic pigment but no nodules; a mild amount of anthracotic pigment which was mostly subpleural and around blood vessels; an acute vascular congestion in focal areas and in some alveoli macrophages containing

hemosiderin; and a mild amount of centrilobular emphysema. In the left lung Dr. Caffrey found a section of main stem bronchus which showed ulceration of the mucosa with inflammatory cells with a normal ratio of mucous to serous glands; acute bronchopneumonia; a mild amount of anthracotic pigment subpleurally and around blood vessels; hemosiderin pigment in macrophages within a number of alveoli; and a mild amount of centrilobular emphysema. Dr. Caffrey concluded that there was no evidence of pulmonary fibrosis, no nodules, and no evidence of asbestosis or complicated pneumoconiosis in either lung.

Based on these findings, Dr. Caffrey concluded that Miner had multiple foci of acute bronchopneumonia and resolving bronchopneumonia; mild centrilobular emphysema; a mild amount of anthracotic pigment within the lungs; a moderate amount of anthracotic pigment in the hilar lymph node tissue; and an acute passive congestion of the lungs. Considering these findings in conjunction with the other medical evidence, 13 years of coal mine employment, and a no smoking history, Dr. Caffrey stated that he was unable to make a diagnosis of CWP or any other occupational pneumoconiosis. He explained that the anthracotic pigment shown on autopsy was insufficient to make a diagnosis of simple CWP as pigment by itself is not synonymous with the disease.

Dr. Caffrey next explained that Miner suffered from several medical diseases of the public in general, including: severe atherosclerosis; a CVA with a large right cerebellar hemisphere CVA; a previous myocardial infarct; diabetes mellitus; and a previous bilateral BK amputation of his lower extremities. Prior to his death, Miner also developed Methacillin Resistant Staphylococcus Aureus with positive blood and sputum cultures. Dr. Caffrey opined that this condition, in conjunction with Miner's bronchopneumonia, was the immediate cause of death. Furthermore, Dr. Caffrey disagreed with Dr. Combs' conclusion that Miner's respiratory function was severely compromised due to chronic airway disease and anthracosis, because the anthracotic pigment present in the slides does not constitute anthracosis, and would not have caused him any pulmonary disability or compromise his pulmonary function. As a result, Dr. Caffrey concluded that Miner's prior dust exposure did not cause, contribute to, or hasten his death.

Dr. Jerrold L. Abraham, a pathologist, submitted a report on August 31, 2004. (CX 1). Dr. Abraham considered 13 years of coal mine employment, Miner's death certificate, Dr. Combs' autopsy report, Dr. Perper's report, Dr. Hansbarger's report, and his own review of the autopsy slides. His microscopic evaluation of the slides revealed abundant accumulation of macrophages containing opaque and birefringent dust consistent with a mixed exposure to crystalline materials as well as probable metal-working operations such as welding or grinding, and there was also abundant hemosiderine consistent with congestive heart failure. Dr. Abraham also identified areas showing acute inflammation consistent with Miner's recent pneumonia diagnosis, as well as evidence of increased mucus gland size, increased goblet cells in the airways, and a mild degree of centrilobular emphysema. Based on these findings, Dr. Abraham stated that the slides showed evidence of mixed pneumoconiosis consistent with occupational exposures to dust including possible coal dust. He also opined that these occupational exposures would certainly have compromised Miner's lung function and reduced his ability to oxygenate blood. He concluded that this decreased lung capacity would have hastened Miner's death in making him more sensitive to cardiovascular disease and pneumonia. Dr. Abraham added that

he was unable to evaluate the precise extent to which pneumoconiosis was a factor in Miner's death, but noted that it would have been a significant contributing factor.

Concerning Dr. Perper's report, Dr. Abraham noted disagreement with the finding of birefringent crystals consistent with silica. He criticized Dr. Perper's report by stating:

> Dr. Perper notes that he did not see any birefringent crystals. This is in striking distinction to my observations of the slides with the standard polarized light optics in my microscope, which I have been using for over 20 years. Birefringent crystals consistent with silica were noted. There was accumulation of macrophages in the interstitium of the lung up to the size visible in the slides without a microscope. I do not know what Dr. Perper means by "anthracotic deposition, slight in perivascular and peribronchial areas, none reaching dimensions of macules." As far as I am aware, there is no minimum dimension for a macule. A macule means an accumulation of dust and macrophages, without much fibrosis (i.e. a 'spot'). The areas of Mr. Gardner's lung that show accumulation of macrophages containing dust related to his occupation, without much fibrosis, are certainly macules in my experience. Dr. Perper did describe areas of interstitial fibrosis in the lung, but he did not observe the birefringent silica crystals and other crystals, which are readily evident with light microscopic examination in the lymph nodes.

Dr. Bruce Broudy, an internist, pulmonologist, and B-reader, submitted a medical evidence review on July 15, 2004. (EX 2). With exception of Dr. Abraham's report, Dr. Broudy reviewed and summarized all of the medical evidence of record. Based on these reports, Dr. Broudy opined that Miner did not have pneumoconiosis. Specifically, he noted that anthracotic pigment is not a diagnostic of CWP or silicosis, the absence of valid lung function studies, that the ABG studies showed only mild to moderate hypoxemia, and the fact that Miner's medical problems developed 20 years after he stopped working in the mines. In addition, Dr. Broudy emphasized Miner's complications of diabetes resulting in renal failure and requiring dialysis, and his vascular disease, but he explained that these impairments were not due to occupational exposure to coal dust. Dr. Broudy concluded that Miner did not suffer a pulmonary disability caused by coal mine employment.

Dr. Broudy was deposed by the Employer on August 6, 2006, when he repeated the findings of his earlier written report. (EX 4). He added that it appeared that Miner died from end-stage renal disease, and opined that the inhalation of coal, rock, or sand dust did not cause, contribute to, or hasten Miner's death. Dr. Broudy explained that since there was no evidence of progressive, massive fibrosis which could lead to death from pneumoconiosis, and based on the absence of pathological or radiographic evidence of CWP or silicosis, in his judgment, Miner's death would have occurred at the time that it did in the manner that it did regardless of whether he was ever employed in the coal-mining industry. On cross-examination, Dr. Broudy explained that he based his cause of death diagnosis on the death certificate.

Dr. Gregory Fino, an internist, pulmonologist, and B-reader, submitted a medical evidence review July 29, 2004. (DX 3). With exception of Dr. Abraham's report, Dr. Fino

reviewed and summarized all of the medical evidence of record. Based on this review, Dr. Fino concluded that there was no objective evidence to support a diagnosis of a coal mine dust related pulmonary condition, COPD of any etiology, or any lung condition. Furthermore, he opined that there was no objective evidence to suggest that lung disease, regardless of cause, played a role in Miner's death. In fact, Dr. Fino stated that based on his review of the evidence, Miner did not suffer from a respiratory impairment. Dr. Fino next opined that Miner's death was the result of a large stroke complicated by kidney disease, and that lung disease played no role. Even assuming that Miner suffered from CWP, Dr. Fino continued to hold the belief that there was no respiratory impairment in this case.

Dr. Fino was deposed on September 7, 2004, when he repeated the findings of his earlier written report. (EX 5). Dr. Fino reiterated that Miner died as the result of his stroke, which he opined caused the loss of consciousness and an inability for his brain to tell his lungs to breath. Also, he noted Miner's diabetes and the hemodialysis as contributory factors, but explained that these were diseases of the general population, and thus, unrelated to coal mine employment. Turning to the diagnosis of COPD, Dr. Fino stated that there was not any objective evidence in the record to support such a diagnosis, namely, there were no valid PFTs, none of the medication lists include lung disease treatment drugs, there is no record of treatment for any lung disease prior to Miner's death, and none of the pathology reports discuss findings that would account for obstructive lung disease in an significant way.

In addition to the evidence he reviewed for his previous report, Dr. Fino also had an opportunity to review Dr. Abraham's report prior to this deposition. Dr. Fino disagreed with Dr. Abraham's diagnosis, noting that the report did not include any description of coal macules or fibrosis. He explained that the mere presence of dust in the lungs absent a tissue reaction with macules and pulmonary fibrosis is not consistent with CWP. Dr. Fino next disagreed with the statement that the various exposures would have compromised lung functions by reiterating that there is no objective evidence that lung function was compromised. He further explained that the only way to show lung impairment is by a valid lung function study, which was not present in this case. In addition, he disagreed with Dr. Abraham's opinion on Miner's reduced ability to oxygenate blood, noting that while the 1996 ABG, the only one conducted on room air,[21] showed minimal hypoxemia which he believed could be explained by a repeatedly diagnosed enlarged heart and the dialysis treatment. Dr. Fino opined that Dr. Abraham's conclusion that Miner's decreased lung capacity would have hastened death was an incorrect statement, noting that numerous studies failed to reveal a higher incident of or a higher rate of death due to cardiovascular disease or pneumonia. Dr. Fino concluded that coal dust exposure would have

[21] Dr. Fino explained that all of the ABG studies, with exception of the 1996 test, were conducted during treatment when Miner was being administered oxygen for his underlying heart problems. As a result, he recommended that these studies not be used for impairment diagnosis or to determine chronic oxygen capacity. With regard to all of the June 2000 ABG studies, the regulations support Dr. Fino's opinion:

> If one or more blood-gas studies producing results which meet the appropriate table in Appendix C is administered during a hospitalization which ends in the miner's death, then any such study must be accompanied by a physician's report establishing that the test results were produced by a chronic respiratory or pulmonary condition. Failure to produce such a report will prevent reliance on the blood-gas study as evidence that the miner was totally disabled at death.

§718.105(d).

played no role in the development of a "large stroke," pneumonia, low blood-sugar, or end-stage kidney disease and it was these conditions that led to his death. Finally, even if Miner suffered from simple CWP, Dr. Fino concluded that this condition would not have hastened death.

On cross-examination, Dr. Fino was asked whether Miner's centrilobular emphysema diagnosed by microscopic evaluation was a form of COPD. He responded that this condition was diagnosed by some of the pathologists, but not all, and that if it results in obstructive changes on the PFT then it can constitute COPD. Dr. Fino noted that the minimal amount of centrilobular or centriacinar emphysema noted by the pathologists was not enough to result in any functional defect, and that it was about the same amount that one would expect to find in a normal population of nonsmokers or non-coal miners.

Smoking History

Dr. Fritzhand reported that Miner smoked three to four cigars per day from 1940 until 1943. (DX 30-42). Otherwise, the record includes no reported smoking history. Therefore, I find that Miner smoked three to four cigars per day for three years, but did not smoke for the last 57 years of his life.

DISCUSSION AND APPLICABLE LAW

Mrs. Gardner filed her survivor's claim on August 22, 2000. (DX 2). Entitlement to benefits must be established under the regulatory criteria at Part 718. *See Neeley v. Director, OWCP*, 11 B.L.R. 1-85 (1988). The Act provides that benefits are provided to eligible survivors of a miner whose death was due to pneumoconiosis. § 718.205(a). In order to receive benefits, the claimant must prove that:

1). The miner had pneumoconiosis;

2). The miner's pneumoconiosis arose out of coal mine employment; and

3). The miner's death was due to pneumoconiosis.

§§ 718.205(a). Failure to establish any of these elements by a preponderance of the evidence precludes entitlement. *See Anderson v. Valley Camp of Utah, Inc.*, 12 B.L.R. 1-111, 1-112 (1989); *Trent v. Director, OWCP*, 11 B.L.R. 1-26, 1-27 (1987). However, the Board has held that, in a survivor's claim under Part 718, the administrative law judge must make a threshold determination as to the existence of pneumoconiosis under 20 C.F.R. § 718.202(a) prior to considering whether the miner's death was due to pneumoconiosis. *Trumbo v. Reading Anthracite Co.*, 17 B.L.R. 1-85 (1993).

Pneumoconiosis

In establishing entitlement to benefits, Claimant must initially prove the existence of pneumoconiosis under § 718.202. Claimant has the burden of proving the existence of pneumoconiosis, as well as every element of entitlement, by a preponderance of the evidence.

See Director, OWCP v. Greenwich Collieries, 512 U.S. 267 (1994). Pneumoconiosis is defined by the regulations:

> For the purpose of the Act, "pneumoconiosis" means a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. This definition includes both medical, or "clinical" pneumoconiosis and statutory, or "legal" pneumoconiosis.
>
> (1) *Clinical Pneumoconiosis*. "Clinical pneumoconiosis" consists of those diseases recognized by the medical community as pneumoconiosis, i.e., conditions characterized by permanent deposition of substantial amounts of particulate matter in the lungs and the fibrotic reaction of the lung tissue to that deposition caused by dust exposure in coal mine employment. This definition includes, but is not limited to, coal workers' pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, silicosis or silicotuberculosis, arising out of coal mine employment.
>
> (2) *Legal Pneumoconiosis*. "Legal pneumoconiosis" includes any chronic lung disease or impairment and its sequelae arising out of coal mine employment. This definition includes, but is not limited to, any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment.

Section 718.201(a).

Section 718.202(a) sets forth four methods for determining the existence of pneumoconiosis.

(1) Under § 718.202(a)(1), a finding that pneumoconiosis exists may be based upon x-ray evidence. The x-ray reports found in the treatment records are not in compliance with the quality standards of §718.102 and Appendix A to Part 718 and may not be considered under § 718.202(a)(1). The remaining x-ray evidence includes seven interpretations of two chest films and one quality only reading. All of these readings were either found to be non-qualifying or totally negative for the presence of pneumoconiosis. In addition the only physician of record to hold advanced credentials, Dr. West, found the February 28, 1996 film to be completely negative. Therefore, I find that Claimant has failed to establish the existence of pneumoconiosis through x-ray evidence under subsection (a)(1).

(2) Under § 718.202(a)(2), a determination that pneumoconiosis is present may be based upon biopsy or autopsy evidence. A diagnoses of pulmonary anthracosis has been held to be the equivalent of a diagnosis of pneumoconiosis. *Dagnan v. Black Diamond Coal Mining Co.*, 994 F.2d 1536 (11th Cir. 1993); *Bueno v. Director*, OWCP, 7 B.L.R. 1-337 (1984); *Smith v. Island Creek Coal Co.*, 2 B.L.R. 1-1178 (1980); *Luketich v. Bethlehem Mines Corp.*, 2 B.L.R. 1-393 (1979). The Sixth Circuit held that the administrative law judge must also consider biopsy evidence which indicates the presence of anthracotic pigment. *Lykins v. Director, OWCP*, 819 F.2d 146 (6th Cir. 1987). However, in *Griffith v. Director, OWCP*, 49 F.3d 184 (6th Cir. 1995), the Sixth Circuit held that a finding of pigmentation described as "yellow-black consistent with

coal pigment" was insufficient, standing alone, to support a finding of pneumoconiosis. By unpublished decision in *Taylor v. Director, OWCP*, BRB No. 01-0837 BLA (July 30, 2002) (unpublished), the Board noted that a physician concluded, on autopsy, that no coal workers' pneumoconiosis was present and, yet he also stated that there was "minimal anthracosis in the mediastinal lymph nodes." As a result, the Board remanded the case to the ALJ to determine whether the legal definition of pneumoconiosis at 20 C.F.R. §718.201 (2001), which includes anthracosis, was satisfied. The Board held that "anthracosis found in lymph nodes may be sufficient to establish the existence of pneumoconiosis."

The record in this case includes a prosector's autopsy report and four physician reviews of the resulting slides. Drs. Casey and Fino also considered the findings expressed by the physicians who reviewed the slides. It is reasonable to assign greater weight to the opinion of the physician who performs the autopsy over the opinions of others who review his or her findings without reviewing the slides. *Terlip v. Director, OWCP*, 8 B.L.R. 1-363 (1985); *Fetterman v. Director, OWCP*, 7 B.L.R. 1-688 (1985). Therefore, since Drs. Casey and Fino did not review the slides themselves, I accord their opinions lesser weight under subsection (a)(2) than I do the opinions of physicians that actually reviewed the slides.

In the autopsy report, Dr. Combs identified evidence of chronic airway disease, but she did not identify a cause of this condition. She also noted that the alveolar septi were mildly thickened and contained increased numbers of macrophages, many of which were laden with black pigment compatible with carbon particles. Based on these findings, Dr. Combs opined that Miner suffered from bilateral anthracosis and that his severely compromised respiratory function was primarily due to chronic airway disease and anthracosis. I find that Dr. Combs' diagnosis of anthracosis is not sufficient to constitute a finding of clinical pneumoconiosis due to the fact that her report mentions only black pigment compatible with carbon particles. Also, I find that while her diagnosis of chronic airway disease is probative, Dr. Combs has not provided an etiology for this condition, and thus, is not sufficient to constitute a finding of legal pneumoconiosis. The Board has held that autopsy reports must be accorded significant probative value regarding the existence and degree of pneumoconiosis because the pathologist who performs the autopsy sees the entire respiratory system as well as other body systems. *Fetterman v. Director, OWCP*, 7 B.L.R. 1-688, 1-691 (1985). In this case, however, I have found that Dr. Combs has failed to provide an opinion sufficient to prove the existence of either legal or clinical pneumoconiosis. Thus, despite her status as the prosector of Miner's autopsy, I accord Dr. Combs' opinion little weight.

Dr. Hansbarger, a pathologist, stated that the microscopic evidence revealed moderate centrilobular emphysema and COPD, but he did not provide an etiology for this condition. Therefore, I find he has not offered an opinion as to the existence of legal pneumoconiosis.

Turning to clinical pneumoconiosis, Dr. Hansbarger opined that the anthracotic pigment identified in the lungs did not include any reactive fibrosis, and thus, Miner did not suffer from anthracosis. On the other hand, looking at the bronchial lymph nodes, Dr. Hansbarger found that the pigment deposition contained reactive fibrosis. Based on this conclusion, he diagnosed anthracosilicosis of the bronchial lymph nodes, but explained that since this condition was not found in the lung itself, then such a finding does not constitute a diagnosis of CWP. I note,

however, that this conclusion is at odds with the Board's previous determinations. *Taylor*, BRB No. 01-0837 BLA. In addition, anthracosilicosis is one of the diseases that constitutes clinical pneumoconiosis under §718.202(a). Therefore, while Dr. Hansbarger has concluded that the microscopic evidence does not support a finding of pneumoconiosis, I find that his finding of anthracosilicosis of the bronchial lymph nodes is, in fact, a probative finding of clinical pneumoconiosis under subsection (a)(2).

Dr. Perper, a clinical professor of pathology, reviewed the autopsy slides and concluded that Miner suffered from moderately severe centrilobular emphysema with interstitial fibrosis and chronic bronchitis. Dr. Perper opined that the chronic bronchitis and COPD were partially responsible for Miner's death, but they were unrelated to coal dust exposure. An unsupported medical conclusion is not a reasoned diagnosis. *Fuller v. Gibraltar Corp.*, 6 B.L.R. 1-292 (1984). *See also Phillips v. Director, OWCP*, 768 F.2d (8th Cir. 1985); *Smith v. Eastern Coal Co*., 6 B.L.R. 1-1130 (1984); *Duke v. Director, OWCP*, 6 B.L.R. 1-673 (1983)(a report is properly discredited where the physician does not explain how underlying documentation supports his or her diagnosis); *Waxman v. Pittsburgh & Midway Coal Co.,* 4 B.L.R. 1-601 (1982). I find that while Dr. Perper has offered a probative opinion as to the existence of chronic bronchitis and COPD, he has failed to offer any explanation as to why these conditions were "unrelated" to prior occupational exposure to coal dust. Therefore, I find that his conclusion as to the absence of legal pneumoconiosis is not reasoned, and thus, I accord his opinion little probative weight.

Dr. Perper also concluded that Miner does not suffer from clinical pneumoconiosis. He noted that many of the desquamated cells and clusters of macrophages contained black anthracotic pigment; the presence of slight anthracotic deposition in peri-vascular and peri-bronchial areas which did not reach the dimensions of macules; and the absence of pneumoniotic nodules or birefringent silica crystals. In addition, Dr. Perper concluded that the peribronchial lymph nodes showed dense anthracosis but no significant fibrosis or birefringent silica crystals. Despite the presence of anthracotic pigmentation, and based on the absence of pneumocotic macules and nodules, Dr. Perper concluded that Miner does not suffer from pneumoconiosis. According to *Taylor*, however, a finding of "dense anthracosis" in the peribronchial lymph nodes is sufficient to constitute a diagnosis of clinical pneumoconiosis. BRB No. 01-0837 BLA. Furthermore, unlike the anthracotic pigment found in the lung tissue, Dr. Perper did not qualify his finding of anthracosis in the pulmonary lymph nodes as pigmentation only, but instead he stated that it showed "no significant fibrosis or birefringent silica crystals." Based on this wording, it is apparent that Dr. Perper found fibrosis and birefringent silica crystals but that he did not find a significant amount of this material. Section 718.202(a) includes anthracosis and massive pulmonary fibrosis in the definition of clinical pneumoconiosis, but nowhere does it state that anthracosis must be accompanied by "significant fibrosis or birefringent silica crystals." Therefore, it is apparent that while Dr. Perper has found anthracotic pigment in Miner's lung tissue, which is insufficient for a diagnosis of clinical pneumoconiosis, he has found that the pulmonary lymph nodes contain "dense anthracosis," which does constitute a finding of clinical pneumoconiosis under §718.202(a) and the Board's holding in *Taylor.* As a result, I find Dr. Perper's conclusion that Miner does not suffer from pneumoconiosis to be at odds with his summary of the slide evidence, and thus, accord his finding that Miner does not suffer from clinical pneumoconiosis little probative weight under

subsection (a)(2). On the contrary, I actually find that Dr. Perper's finding of dense anthracosis in the pulmonary lymph nodes constitutes a probative diagnosis of clinical pneumoconiosis.

Dr. Caffrey's review of the autopsy slides revealed that Miner had a mild amount of centrilobular emphysema bilaterally. While he did not specifically state that these conditions were unrelated to coal dust exposure, Dr. Caffrey did conclude that he was unable to make a diagnosis of CWP or any other occupational pneumoconiosis. Also, Dr. Caffrey disagreed with Dr. Combs' conclusion that Miner's respiratory function was severely compromised due to chronic airway disease and anthracosis on the grounds that Miner did not have anthracosis. Considering all of Dr. Caffrey's comments on the issue of legal pneumoconiosis, I find that he has opined that Miner's autopsy revealed bilateral centrilobular emphysema, and he has concluded that this condition is unrelated to coal dust exposure, but he has also failed to provide any explanation for why he excluded coal dust as a possible cause for this condition. Furthermore, while Dr. Caffrey disputed Dr. Combs' impairment conclusion, that statement does not express an opinion one way or the other about whether Miner suffered from chronic airway disease or whether this condition caused a pulmonary impairment. Therefore, I find that Dr. Caffrey has not offered an opinion as to whether Miner suffers from a chronic lung condition, and as a result, his conclusion as to the cause of Miner's centrilobular emphysema is irrelevant to the discussion of legal pneumoconiosis under subsection (a)(2).[22]

Dr. Caffrey also opined that the autopsy slides do not demonstrate the presence of clinical pneumoconiosis. Specifically, Dr. Caffrey identified only a mild amount of anthracotic pigment within the lungs and a moderate amount of anthracotic pigment in the hilar lymph node tissue. He explained that anthracotic pigment, considered by itself, is not synonymous with anthracosis. As Dr. Caffrey based his opinions on the objective evidence that he considered, I find his opinion to be well-documented and well-reasoned. Therefore, I accord his opinion as to the existence of clinical pneumoconiosis probative weight.

Dr. Abraham, a pathologist, reviewed the autopsy slides and concluded that Miner's lung showed evidence of a mild degree of centrilobular emphysema. Dr. Abraham did not offer an opinion to the etiology of this condition. Therefore, I find that he did not diagnose legal pneumoconiosis.

Dr. Abraham next identified an accumulation of macrophages containing opaque and birefringent dust, without much fibrosis, consistent with a mixed exposure to crystalline materials as well as probably metal-working operations. He opined that these accumulations were macules. Dr. Abraham added that these results showed evidence of "mixed pneumoconiosis" consistent with occupational exposures to dust including "possible" coal dust. Dr. Abraham concluded that the birefringent silica crystals and other crystals were also readily evident with microscopic examination in the lymph nodes. An opinion may be given little

[22] Even if Dr. Caffrey had concluded that Miner's centrilobular emphysema rose to the level of COPD, I would accord his opinion only little weight due to his failure to provide any explanation as to why he excluded coal dust as a possible cause for Miner's respiratory condition. Furthermore, I find that this wholesale dismissal, without explanation, of coal dust as a cause significantly colors my analysis of all of his opinions and clearly calls into question his final opinion of Mr. Gardner's respiratory conditions, particularly at the time of his death. This necessarily includes not only his opinion on whether the condition was caused by coal dust exposure, but the question of whether the condition contributed to or hastened Miner's death.

weight if it is equivocal or vague. *Island Creek Coal Co. v. Holdman*, 202 F.3d 873 (6th Cir. 2000) (a physician, who concluded that simple pneumoconiosis "probably" would not disrupt a miner's pulmonary function, was equivocal and insufficient to "rule out" causal nexus as required by 20 C.F.R. §727.203(b)(3)). As Dr. Abraham attributed Miner's mixed pneumoconiosis to "possible" coal dust exposure, I find that he has not provided a definitive diagnosis of legal pneumoconiosis. In fact, his reports appears to be far more certain as to the existence of some sort of metal-working induced pneumoconiosis, but even there, I find his "probable" diagnosis to be equivocal. In addition, while Dr. Abraham specifically identifies macules without much fibrosis in the lungs, birefringent silica crystals and other crystals in the lymph nodes, and presence of silicates, carbonaceous material, crystalline silica, and iron-containing material consistent with exposure to welding or similar occupations in Miner's lungs, he never offers a definite opinion as to the specific type of pneumoconiosis that was found in Miner's lungs, nor does he identify whether his findings in Miner's lymph nodes were consistent with pneumoconiosis. As a result, I find that Dr. Abraham has offered a probative opinion as to the existence of clinical pneumoconiosis, and it is based on the microscopic evidence he considered, but due to the vague and equivocal nature of this opinion, I find that it is entitled only to only some weight.

Concerning legal pneumoconiosis, I have found that Drs. Combs, Hansbarger, Caffrey, and Abraham have not offered an opinion as to whether the autopsy slides demonstrated the presence of legal pneumoconiosis. In addition, I have found that Dr. Perper's opinion that Miner's chronic bronchitis, moderately severe centrilobular emphysema, and COPD were "unrelated" to coal dust exposure to be without support, and thus, entitled to little weight. Based on Dr. Combs' diagnosis of chronic airway disease; Dr. Hansbarger's diagnosis of moderate centrilobular emphysema and COPD; and Dr. Perper's diagnosis of chronic bronchitis, moderately severe centrilobular emphysema, and COPD; it is clear that Miner suffered from some form of chronic lung condition at the time of his death. However, due to the absence of a reasoned opinion as to the etiology of this condition, it is not possible to render a finding as to the existence of legal pneumoconiosis.

Turning to clinical pneumoconiosis, I have found Dr. Hansbarger's report to be probative as to the existence of anthracosilicosis in the bronchial lymph nodes, and Dr. Perper's reports to be probative as to the existence of dense anthracosis in the pulmonary lymph nodes. In addition, I have accorded Dr. Abraham's diagnosis of "mixed pneumoconiosis" in the lung some probative weight. On the other hand, I have found Dr. Caffrey's finding of no clinical pneumoconiosis to be probative. Finally, I have found Dr. Combs' autopsy report to be of little probative weight. Based on the most probative opinions by Drs. Hansbarger, Perper, and Caffrey, it is clear that the preponderance of the microscopic evidence does not support a finding of clinical pneumoconiosis in the lungs proper.

However, whether the pulmonary lymph nodes reveal evidence of the disease presents a much closer call. In the final analysis, I find Dr. Hansbarger's opinions supported by Dr. Perper's findings to be the most convincing, in part, due to Dr. Hansbarger's testimony explaining his understanding of the difference between anthracotic pigment and anthracosis, and then his definitive finding of anthracosilicosis in the pulmonary lymph nodes. Therefore, I accord Dr. Hansberger's diagnosis more probative weight than the other physicians who

performed microscopic evaluations. Thus I find that Miner suffered from anthracosilicosis of the pulmonary lymph nodes which qualifies as clinical pneumoconiosis under subsection (a)(2) and the Boards holding in *Taylor*.

(3) Section 718.202(a)(3) provides that pneumoconiosis may be established if any one of several cited presumptions are found to be applicable. In this case, the presumption of § 718.304 does not apply because there is no evidence in the record of complicated pneumoconiosis. Section 718.305 is not applicable to claims filed after January 1, 1982. Finally, the presumption of § 718.306 is applicable only in a survivor's claim filed prior to June 30, 1982. Therefore, Claimant cannot establish pneumoconiosis under subsection (a)(3).

(4) The fourth and final way in which it is possible to establish the existence of pneumoconiosis under § 718.202 is set forth in subsection (a)(4) which provides in pertinent part:

> A determination of the existence of pneumoconiosis may also be made if a physician, exercising sound medical judgment, notwithstanding a negative x-ray, finds that the miner suffers or suffered from pneumoconiosis as defined in § 718.201. Any such finding shall be based on electrocardiograms, pulmonary function studies, physical performance tests, physical examination, and medical and work histories. Such a finding shall be supported by a reasoned medical opinion.

§ 718.202(a)(4).

This section requires a weighing of all relevant medical evidence to ascertain whether or not the claimant has established the presence of pneumoconiosis by a preponderance of the evidence. Any finding of pneumoconiosis under § 718.202(a)(4) must be based upon objective medical evidence and also be supported by a reasoned medical opinion. A reasoned opinion is one which contains underlying documentation adequate to support the physician's conclusions. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19, 1-22 (1987). Proper documentation exists where the physician sets forth the clinical findings, observations, facts, and other data on which he bases his diagnosis. *Oggero v. Director, OWCP*, 7 B.L.R. 1-860 (1985).

The reports by Drs. Hansbarger, Perper, Caffrey, and Abraham were previously discussed in subsection (a)(2). While their analysis under subsection (a)(2) dealt primarily with their reviews of the autopsy slide evidence, I note that each of these physicians also reviewed large portions of the medical evidence of record. Upon review of their complete reports, I find that my findings with regard to their clinical and legal pneumoconiosis conclusions remain unchanged. Specifically, I find that the preponderance of these reports support a finding that Claimant suffered from a chronic lung condition, but there is not a reasoned report as to the etiology of this condition. In addition, based primarily on the microscopic review by Dr. Hansbarger, supported by the findings by Dr. Perper, I find that the preponderance of the evidence supports a diagnosis of clinical pneumoconiosis in the form of anthracosilicosis of the pulmonary lymph nodes.

The record in this case also includes a number of treatment records, and medical reports by Drs. Fritzhand, Casey, Fino, and Broudy. Dr. Fritzhand considered Miner's employment history, smoking history, an insignificant physical examination, a negative chest x-ray, an invalid PFT, and an ABG, and concluded that Miner suffered from ASHD. In addition, while he did not specifically identify any pulmonary abnormalities, he did conclude that Miner did not suffer from a pulmonary impairment. Since Dr. Fritzhand submitted Miner to the entire regimen of pulmonary testing and definitively stated that Miner does not suffer from a pulmonary impairment, I do not find that his failure to specifically exclude the possibility of clinical or legal pneumoconiosis to equate to silence on the issue. On the contrary, it is apparent that Dr. Fritzhand reviewed the objective evidence before him and determined that it did not support a finding of pneumoconiosis. Therefore I find his report to be a well-reasoned and well-documented finding of the absence of the disease, and thus, accord his opinion probative weight.

Dr. Casey was Miner's treating physician, and in addition to treatment notes, she also completed the death certificate and submitted an opinion letter. Dr. Casey's treatment notes include a May 15, 2000 diagnosis of chronic bronchitis, a June 22, 2000 diagnosis of respiratory distress with hypoxia and COPD, and a June 25, 2000 diagnosis of COPD. The treatment record also includes a diagnosis of bronchitis on January 13, 1999 by Dr. Shapiro, and a finding of severe respiratory failure due to volume overload and COPD by Dr. Bhagrath. While these COPD diagnoses were all supported by examination and testing results, and are consistent with my conclusions based on the autopsy slides, I note that at no point in the treatment records does any physician attribute these pulmonary problems to exposure to coal dust, or even note that Mr. Gardner previously worked in the mines. In fact, the first time Dr. Casey ever mentioned pneumoconiosis of any form was on the August 7, 2000 death certificate. I find that her 2001 letter stating that Miner suffered from COPD secondary to CWP clarifies that Dr. Casey was diagnosing legal pneumoconiosis, but with exception of the attached death certificate and Dr. Combs' autopsy report, she provides no explanation of what evidence she considered in reaching this conclusion. I further note that Dr. Combs' autopsy report does not provide any support to a finding of legal pneumoconiosis. As a result, considering all of the submissions by Dr. Casey, I find no objective support for her contention that Miner's COPD was caused by coal mine employment and no mention that she ever considered any specific coal mine employment. *See Duke v. Director, OWCP*, 6 B.L.R. 1-673 (1983)(a report is properly discredited where the physician does not explain how underlying documentation supports his or her diagnosis); *Cosaltar v. Mathies Coal Co.*, 6 B.L.R. 1-1182 (1984)(a physician's report may be rejected where the basis for the physician's opinion cannot be determined); *Worhach v. Director, OWCP*, 17 B.L.R. 1-105 (1993)(per curiam)(it is proper for an ALJ to discredit a medical opinion based on an inaccurate length of coal mine employment). As a result, I find that Dr. Casey's diagnosis of legal pneumoconiosis not to be well-reasoned or well-documented. Therefore, despite her status as Miner's treating physician, I accord her opinion little weight.

Dr. Broudy, an internist, pulmonologist, and B-reader, concluded that Miner did not suffer from pneumoconiosis. This opinion was based on the pathology findings of anthracotic pigment only; the absence of valid lung function studies; the fact that the ABG studies showed only mild to moderate hypoxemia; and the fact that Miner's medical problems developed 20 years after he stopped working in the mines. I note that his statement regarding anthracosis is inconsistent with the findings by Dr. Hansbarger. Since I found Dr. Hansbarger's report to be

the most probative under subsection (a)(2), and since Dr. Hansbarger actually reviewed the autopsy slides, I find that the weight of Dr. Broudy's contrary opinion as to the existence of clinical pneumoconiosis is greatly diminished. Furthermore, while Dr. Broudy's conclusions with regard to the ABG and PFT studies are consistent with the evidence he considered, he fails to provide any explanation as to why he discounted the diagnoses of COPD, including centrilobular emphysema and chronic bronchitis found by the pathology reports and the examination reports within the treatment records. As a result, I find that by totally neglecting to address significant aspects of the record before him, the weight of Dr. Broudy conclusions is greatly diminished. Therefore, I find that due to Dr. Broudy's selective consideration of the record before him, his conclusions as to the absence of pneumoconiosis are not well-reasoned or well-documented. Thus, I accord his opinions little weight.

Dr. Fino, and internist, pulmonologist, and B-reader, reviewed the medical evidence of record and concluded that there was no objective evidence to support a diagnosis of a coal dust related pulmonary condition or COPD of any etiology. While I do find convincing his argument that none of Miner's prescribed medications were for the purpose of treating lung disease, I also find that Dr. Fino's opinions suffer from the same deficiencies as those by Dr. Broudy. Specifically, while Dr. Fino is correct in noting that the ABG and PFT studies of record do not clearly support a finding of legal pneumoconiosis, he fails to consider the diagnoses of COPD, many of which were diagnosed by means of physical examination. He has also failed to consider the pathology evidence which I have found to be probative for the existence of COPD. As a result, for the same reasons that I rejected Dr. Broudy's opinion, I now find that Dr. Fino's conclusions as to the absence of pneumoconiosis are not well-reasoned or well documented. Thus, I accord his opinions little weight.

It is clear from the treatment records and the pathology evidence that Miner suffered from COPD, but there are no well-reasoned and well-documented medical reports of record that provide a cause of this lung condition. Therefore, I conclude that Claimant has failed to prove by a preponderance of the evidence, that Miner suffered from legal pneumoconiosis pursuant to subsection (a)(4). Turning to clinical pneumoconiosis, with exception of those reports previously analyzed under subsection (a)(2), there are no reasoned or documented medical opinions exclusive to subsection (a)(4) finding that Miner suffers from the disease. Therefore, I find that Claimant has failed to prove that Miner suffered from either clinical or legal pneumoconiosis under subsection (a)(4).

Claimant has failed to establish the presence of either legal or clinical pneumoconiosis under subsections (a)(1), (3) and (4), but has proven by a preponderance of the autopsy evidence under subsection (a)(2) that he suffers from clinical pneumoconiosis. Therefore, I find that Claimant has shown the presence of pneumoconiosis by a preponderance of the evidence under §718.202 (a).

Arising out of Coal Mine Employment

In order to be eligible for benefits under the Act, Claimant must prove that pneumoconiosis arose, at least in part, out of Miner's coal mine employment. § 718.203(a). For a miner who suffers from pneumoconiosis and was employed for ten or more years in one or more coal mines, it is presumed that his pneumoconiosis arose out of his coal mine employment.

Id. As I have found that Claimant has established at least 13 years of coal mine employment, I find that Miner's pneumoconiosis arose out of his coal mine employment in accordance with the rebuttable presumption set forth in § 718.203(b), to which no contrary evidence was offered by Employer.

Death Due to Pneumoconiosis

Having established that Miner suffered from pneumoconiosis arising out of coal mine employment, Mrs. Gardner is now required to prove that Miner's death was due to pneumoconiosis in order to be entitled to benefits. Subsection 718.205(c) applies to survivor's claims filed on or after January 1, 1982 and provides that an eligible survivor will be entitled to benefits if any of the following criteria are met:

1. Where competent medical evidence establishes that pneumoconiosis was the cause of the Miner's death, or

2. Where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or where death was caused by complications of pneumoconiosis, or

3. Where the presumption set forth in § 718.304 (evidence of complicated pneumoconiosis) is applicable.

20 C.F.R. § 718.205(c).

Pneumoconiosis is a "substantially contributing cause" of a miner's death if it hastens the miner's death. § 718.205(c)(5). The presumption set forth in § 718.304 is not applicable because Claimant has not established the presence of complicated pneumoconiosis. Therefore, in order for Claimant to be entitled to benefits, she must show that pneumoconiosis was the direct cause of Miner's death or that pneumoconiosis hastened Miner's death.

A death certificate, in and of itself, is an unreliable report of the miner's condition and it is error for an administrative law judge to accept conclusions contained in such a certificate where the record provides no indication that the individual signing the death certificate possessed any relevant qualifications or personal knowledge of the miner from which to assess the cause of death. *Smith v. Camco Mining, Inc.*. 13 B.L.R. 1-17 (1989); *Addison v. Director, OWCP*, 11 B.L.R. 1-68 (1988).

The record includes two opinions stating that Miner's death was due to pneumoconiosis. Dr. Casey listed on the death certificate that Miner's death was due, in part, to COPD and mild bilateral pneumoconiosis. In her letter she clarified that Miner's COPD was secondary to CWP and that the autopsy report revealed bilateral anthracosis. I have found that Dr. Casey's legal pneumoconiosis analysis lacks reference to any well-reasoned or well-documented opinions included in the record which would link Miner's chronic lung condition to coal dust exposure. As a result, Dr. Casey's conclusion that Miner's death was due to legal pneumoconiosis is also without sufficient reason. Next, Dr. Casey stated that Miner suffered from bilateral anthracosis,

so her letter could also be construed as an opinion that his death was due to clinical pneumoconiosis. However, since her clinical pneumoconiosis analysis appears to be based solely on Dr. Combs' autopsy report, and since I have found Dr. Combs report to be insufficient proof of the existence of clinical pneumoconiosis, I find that Dr. Casey's conclusions are not sufficiently well-reasoned. Furthermore, even if I had found Dr. Casey's clinical or legal pneumoconiosis diagnoses to be well-reasoned and well documented, I would continue to negate her cause of death analysis due to inconsistencies in her documentation. On the June 26 discharge report, Dr. Casey listed 28 distinct discharge diagnoses but at no point does she mention coal dust exposure, or pneumoconiosis of any variety. However, on the subsequent death certificate Dr. Casey lists only four causes, including mild bilateral pneumoconiosis, and one contributing cause. This failure to explain why she added pneumoconiosis as a cause of death in her subsequent report further diminishes the weight of her opinion. Therefore, I must accord Dr. Casey's cause of death determination little weight.

Dr. Abraham also concluded that Miner's death was due to clinical pneumoconiosis. Specifically, he stated that Miner's occupational exposures would certainly have compromised Miner's lung function and reduced his ability to oxygenate blood, and this decreased lung capacity would have hastened Miner's death by making him more sensitive to cardiovascular disease and pneumonia. In addition, Dr. Abraham noted that he was not able to determine the extent to which pneumoconiosis was a factor in Miner's death, but it was a significant contributing factor.

A medical opinion based upon generalities, rather than specifically focusing upon the miner's condition, is insufficient. *Knizer v. Bethlehem Mines Corp.*, 8 B.L.R. 1-5 (1985). I find that Dr. Abraham does not provide any evidence to support his conclusion that clinical pneumoconiosis hastened or contributed to this miner's death, but instead, has provided a general, unsupported conclusion. Specifically, I find that Dr. Abraham's statement that unspecified[23] occupational exposures "would certainly" have led to death is not equivalent to an objectively supported explanation as to how coal dust exposure actually led to death.[24] Therefore, I accord Dr. Abraham's generalized conclusion little weight.

I have found none of the medical opinions attributing Miner's death to pneumoconiosis to be well-reasoned and well-documented. Therefore, I find that Claimant has failed to established that Miner's death was due to pneumoconiosis arising out of coal mine employment under § 718.205(c).

Entitlement

While Claimant, Reba Gardner, has shown that her husband, Daniel Gardner, suffered from pneumoconiosis arising out of coal mine employment, she has failed to prove, by a

[24] I find that Dr. Fino's criticisms of Dr. Abraham's report to be on point. First, Dr. Fino notes that there is no objective evidence in the record that Miner's lung function was compromised due to the fact that the PFTs were invalid. Second, Dr. Fino correctly explained that the evidence was insufficient to prove Miner's inability to oxygenate blood and that the minimal hypoxemia values found in 1996 were explainable by Miner's repeatedly diagnosed enlarged heart and ongoing dialysis treatment. Therefore, I find that Dr. Fino's criticisms of Dr. Abraham's opinion as to cause of death to be better reasoned and supported by the objective evidence of record.

preponderance of the evidence that his death was due to the disease. Therefore, I find that Mrs. Gardner is not entitled to benefits under the Act.

Attorney's Fees

An award of attorney's fees is permitted only in cases in which the claimant is found to be entitled to benefits under the Act. Because benefits are not awarded in this case, the Act prohibits the charging of any fee to the Claimant for the representation and services rendered in pursuit of the claim.

ORDER

IT IS ORDERED that the claim of Reba Gardner for benefits under the Act is hereby **DENIED.**

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THOMAS F. PHALEN, JR.
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).